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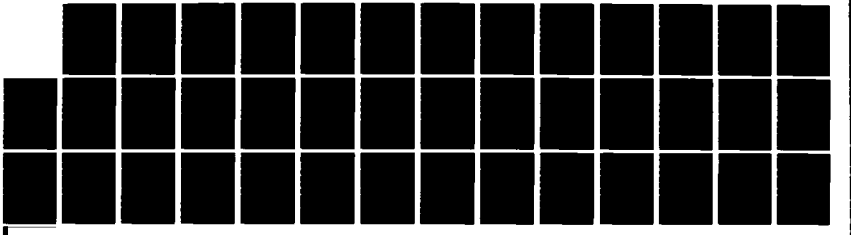

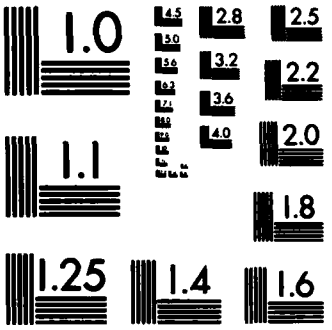

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Efforts during this reporting period have been directed in three areas: 1) Examination of regional intrapleural pressure changes during +Gz stress in the pig; (2) Development of an inexpensive assist/control, volume limited animal ventilator; and (3) Determining the influence of chest wall motion on gas exchange during mechanical ventilation in dogs.

Studies assessing regional intrapleural pressure changes during +Gz stress in the dog were repeated in similar sized pigs to determine the role of chest wall mechanics in determining these changes. Unlike the earlier dog

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[illegible] the rib cage was detected in which [illegible] intrapleural pressure remained relatively constant during +Gz stress. At all levels measured, pleural pressure became more negative with increasing +Gz stress. When the G-suit abdominal bladder was used, increases in regional intrapleural pressure greater than those seen in analogous dog experiments were observed. These results imply that, as the chest wall becomes less compliant, the degree of lung compression attributable to +Gz stress without G-suit application should deminish. In another series of experiments, gas exchange during assisted and controlled ventilation were compared in an attempt to ascertain whether an active effort by chest wall muscles coordinated with inspiration can influence gas exchange. The data obtained indicate that an inspiratory muscular effort enhances gas exchange. Measured gas exchange parameters suggest that this enhancement is the result of a redistribution of perfusion rather than a redistribution of ventilation.

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INTERACTION OF ANTI-G MEASURES AND CHEST WALL MECHANICS IN DETERMININC GAS EXCHANGE

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ANIMAL USE STATEMENT:

Care has been taken in these studies to ensure that all animal experimentation complies with all federal animal welfare regulations and the "Guide for the Care and Use of Laboratory Animals".

High sustained gravitational stress (HSG), such as that experienced by pilots of high performance aircraft, affects cardiovascular and respiratory function adversely (Burton et al., 1974). Cardiovascular function is compromised because of changes in hydrostatic relationships caused by the increased G. Similar mechanisms influence distribution of ventilation and perfusion (Bryan et al., 1966; Jones et al., 1969; Glaister, 1970; von Nieding and Krekeler, 1973). In addition, the HSG may alter chest wall mechanics (Hershgold, 1960) and impair gas exchange. A number of protective measures are presently employed in an attempt to restore normal arterial blood pressure and, thus, increase pilot tolerance to high sustained gravitational forces. Some of these measures (e.g. anti-G suits) have been associated with additional detriment to pulmonary gas exchange (Barr, 1962; Nolan et al., 1963; Hyde et al., 1963), whereas others (e.g. positive pressure breathing) may enhance pulmonary gas exchange under HSG conditions. Several questions relevant to HSG tolerance must be addressed if more effective protective measures are to be developed:

1. To what extent do commonly used protective measures enhance or impair pulmonary gas exchange?

2. What is the time course of any gas exchange detriment resulting from use of protective devices (e.g. anti-G suits) during HSG?

3. Is there a cumulative effect associated with gas exchange detriment resulting from use of protective devices?

4. By what means can these measures be modified to optimize gas exchange during HSG?

This project has focused on these questions. In previous years, studies have dealt primarily with the influence of +Gz stress on chest

wall-pulmonary mechanics and gas exchange in dogs. Questions related to the influence of G-suit inflation on the intrapleural pressure gradient and the time course of gas exchange detriment during HSG have been addressed. In addition, the first steps in extending these studies to an animal whose chest wall compliance more closely approximates that of man (i.e., the pig) were begun. During the last twelve months, these studies have continued, and new studies were begun aimed at better understanding of the chest wall-gas exchange interaction. This report will focus on three such efforts: 1) Influence of HSG and G-suit abdominal bladder inflation on the intrapleural pressure gradient in pigs; 2) design of a new animal ventilator allowing delivery of animal triggered breaths; 3) influence of a co-ordinated inspiratory effort on gas exchange during mechanical ventilation in dogs.

I. INFLUENCE OF +Gz AND G-SUIT INFLATION ON THE INTRAPLEURAL PRESSURE GRADIENT IN PIGS

The vertical intrapleural pressure gradient has been attributed to the influence of gravity acting on the lung (Krueger et al., 1961; Vawter et al., 1975) and to lung deformation within the chest wall (Agostoni and D'Angelo, 1971; Agostoni et al., 1970). Although reports have appeared describing the distribution of intrapleural pressure along the vector of gravitational stress in dogs at 0Gz (supine) and +1Gz (head-up posture) (Hoppin et al., 1969; Krueger et al., 1961; McMahon et al., 1969), none have examined the influence of high +Gz stress on this variable. Wood et al. (1963) measured intrapleural fluid pressure at various sites during +Gx (anterior to posterior) stress. However, these authors did not consider the +Gz (cranial to caudal) vector nor did they examine the influence of a G-suit on the chest wall-lung system.

In earlier studies on this project, we measured regional intrapleural pressure changes in dogs exposed to up to +5Gz with and without G-suit abdominal bladder inflation (Modell, 1981). Results from those studies indicated that, without G-suit inflation, intrapleural pressure above the level of the 7th-8th intercostal space became more negative with increasing +Gz stress, while below the 7th-8th intercostal space level, intrapleural pressure became more positive with increasing +Gz stress. This suggests that, in the dog, a region exists where intrapleural pressure remains relatively constant with +Gz stress. We interpreted this region as reflecting the transition between a relatively stiff upper chest wall and the more flexible lower chest wall.

The compliance of the human chest wall is less than that of the dog and closer to that of the pig (Attinger and Cahill, 1960). To gain a

better appreciation of the importance of this variable on the intrapleural pressure gradient during HSC, studies similar to those in the dog were conducted in the pig.

Methods

Ten Duroc-Yorkshire pigs weighing 20.4 $\pm$ 1.3 Kg were anesthetized with 18 mg/Kg ketamine hydrochloride and 2 mg/Kg Xylazine adminstered intramuscularly. An external jugular vein was cannulated for supplemental anesthesia administration (Pentobarbital Sodium), a tracheostomy was performed, and a Millar catheter-tip pressure transducer was introduced through the carotid artery and advanced to the thoracic aorta level for arterial blood pressure monitoring. Balloon covered, stainless steel cannulae were placed in three intercostal spaces ranging from the third to tenth intercostal space. The cannulae were constructed from 3 inch, 15 guage needles. The needles were blunted and bent at a right angle 4 cm from the tip, and 24 side holes were ground along the length of the cannula. The cannula was cut and "hinged" with a small piece of silastic tubing 2 cm from the tip, and a thin finger cot was placed over the 4 cm length and fastened at the bend. Anchors were attached to the needle hub so that the cannula could be secured with suture to the skin.

During insertion of the cannulae, the animal was ventilated mechanically, and 10-15 cm H_2O end-expiratory pressure was imposed to insure that a pneumothorax was not created. The cannulae were placed in the same vertical plane along the ventral third of the lateral surface of the rib cage. They were directed along the intercostal space with the tips facing dorsally. Purse string sutures through at least one muscle layer and the skin were tightened around the cannulae to prevent

air leakage into the intrapleural space during the experiment. Mechanical ventilation and positive end-expiratory pressure were removed, and the animal was allowed to breathe spontaneously.

When all pleural pressure monitoring sites had been established, a standard G-suit abdominal bladder (CSU-12/P) was placed around the animal's abdomen. Care was taken to ensure that the G-suit was positioned well below the level of monitoring cannulae. The pig was placed supine on the animal end of one of two centrifuges (Wright-Patterson AFB or USAFSAM). Imposed +Gz stress consisted of 40 second exposures to steady +Gz levels of +1 to +5Gz (onset rate = 0.1G/sec). Measurements were made with and without G-suit inflation (standard inflation scheme, 1.5 psi/G starting at approximately +2Gz) using Validyne MP-45 pressure transducers connected through air-filled tubing to the air-filled cannulae. All signals were recorded on a strip-chart recorder, an FM analog magnetic tape recorder, and in the form of digital data points sampled at rates ranging from 2 to 5 samples per second using an Apple][computer system. Between exposures, the animal's lungs were hyperinflated several times to open any atelectatic or airway closure areas.

After data had been collected at each +Gz level with and without G-suit inflation, an overdose of pentobarbital sodium was administered intravenously or the animal was sacrificed with an intravenous injection of saturated KCl. The animal was then exposed to +4 and +5Gz as earlier. The intent of this portion of the protocol was to provide a means by which the effects of the exposure on the passive lung-chest wall system could be separated from any modifying influence of active chest wall muscular tone. These exposures were begun within 5 minutes

post-KCl or pentobarbital overdose and completed with approximately 20 minutes.

At the completion of the experiment, a thoracotomy was performed to confirm the monitoring sites and examine the lungs for any gross damage resulting from the cannulae.

Because data from these studies were to be compared to the earlier dog studies in which open cannulae were used to monitor intrapleural pressure changes, experiments were conducted in 2 dogs and one pig comparing pressure measurements from the two types of cannulae with the animal in the supine posture. In each experiment, cannula pairs (with, without balloon) were compared at one or two intercostal space levels on each side of the animal. For example, in one experiment, one balloon-tipped cannula was placed in the right third intercostal space and one in the left seventh intercostal space. Open cannulae in this experiment were placed in the left third intercostal space and right seventh intercostal space. Pressures were recorded from all cannulae during spontaneous breathing, spontaneous breathing against increased airway resistance, spontaneous breathing with positive end-expiratory pressure, mechanical ventilation, and mechanical ventilation with positive end-expiratory pressure. Pressures compared ranged from approximately -12 cm H_2O to +12 cm H_2O in magnitude. Five cannula pairs were compared. The data were then digitized using an Apple][computer system, and linear regression equations were determined for each cannula pair. The number of points per pair upon which a regression equation was determined ranged from 383 to 1068. Correlation coefficients of the five regressions ranged from 0.899 to 0.996 with the average correlation coefficient being 0.953.

Results

Intrapleural pressure changes from end-expiration (FRC) during the 0 Gz control period were determined at each +Gz level and test condition using the digitized data (2-5 samples/sec). Intrapleural pressure changes were determined at FRC during the 40 second exposure and at each G level during the slow onset phase of the exposure. Mean data obtained from the ten animals are summarized in Tables I-1 and I-2. Data obtained from the third and fourth, fifth and sixth, and eighth and tenth intercostal spaces were pooled so that meaningful statistical analysis could be performed.

The influence of G-stress on regional intrapleural pressure changes are shown in Figures I-1, I-2 and I-3. The change in intrapleural pressure from the the 0 Gz control value at each monitoring site is plotted as a function of +Gz stress for the control state (animal spontaneously breathing and without G-suit) and after the heart had been stopped (control state minus active muscular tone, reflexes, etc.) without the G-suit. At each monitoring site, intrapleural pressure became more negative as the G-stress increased. When active muscular chest wall muscular tone was removed, the rate of change in intrapleural pressure increased.

The influence of G-suit abdominal bladder inflation is also shown in Figures I-1, I-2 and I-3. Only data obtained at +3Gz and above have been plotted since the bladder did not begin to inflate until approximately +2.2Gz, and data obtained at the lower G levels were essentially the same as in the control state. At the +3Gz level, influence of bladder inflation was detected at all monitoring sites. In the non-dependent lung regions, intrapleural pressure approached the control level (Fig. I-1, I-2). At the higher +Gz levels, intrapleural

TABLE I-1. MEAN INTRAPLEURAL PRESSURE CHANGE FROM 0Gz

LIVE PIG

INTERCOSTAL SPACE	N	+Gz 1	2	3	4	5
				WITHOUT G-SUIT		
3 - 4	7	-6.24	-10.64	-13.05	-15.32	-16.56
	(SEM)	0.49	1.21	1.51	1.24	1.95
5 - 6	7	-4.56	-7.69	-8.73	-9.46	-9.93
	(SEM)	0.74	1.20	1.48	1.38	2.54
8 - 10	4	-3.28	-5.77	-6.41	-6.97	-10.80
	(SEM)	0.85	1.17	1.30	1.77	2.32
				WITH G-SUIT		
3 - 4	7	-6.67	-12.15	-4.44	1.96	4.73
	(SEM)	0.47	0.93	1.52	2.18	1.97
5 - 6	7	-5.49	-8.65	-3.64	2.74	6.02
	(SEM)	0.78	1.53	1.61	1.94	2.43
8 - 10	4	-3.30	-5.74	2.00	14.17	20.18
	(SEM)	0.67	0.89	3.60	2.80	3.01

TABLE 1-2. MEAN INTERPLEURAL PRESSURE CHANGE FROM 0Gz

DEAD PIG

INTERCOSTAL SPACE	N	+Gz 1	2	3	4	5
				WITHOUT G-SUIT		
3 - 4	7	-5.94	-12.25	-16.81	-18.86	-21.43
	(SEM)	0.82	1.94	3.10	3.76	4.42
5 - 6	6	-5.35	-9.37	-13.50	-16.48	-17.37
	(SEM)	0.93	1.86	2.62	3.13	4.11
8 - 10	4	-3.52	-6.66	-9.30	-11.01	-14.83
	(SEM)	1.06	0.90	1.14	1.28	3.90
				WITH G-SUIT		
3 - 4	4	-9.89	-13.01	-5.08	4.05	8.82
	(SEM)	1.61	0.71	6.07	6.14	8.50
5 - 6	3	-6.04	-8.38	-5.59	6.44	12.63
	(SEM)	2.34	1.92	3.44	1.58	2.37
8 - 10	3	-4.11	-3.50	2.63	19.37	27.88
	(SEM)	1.49	1.69	9.70	7.76	8.06

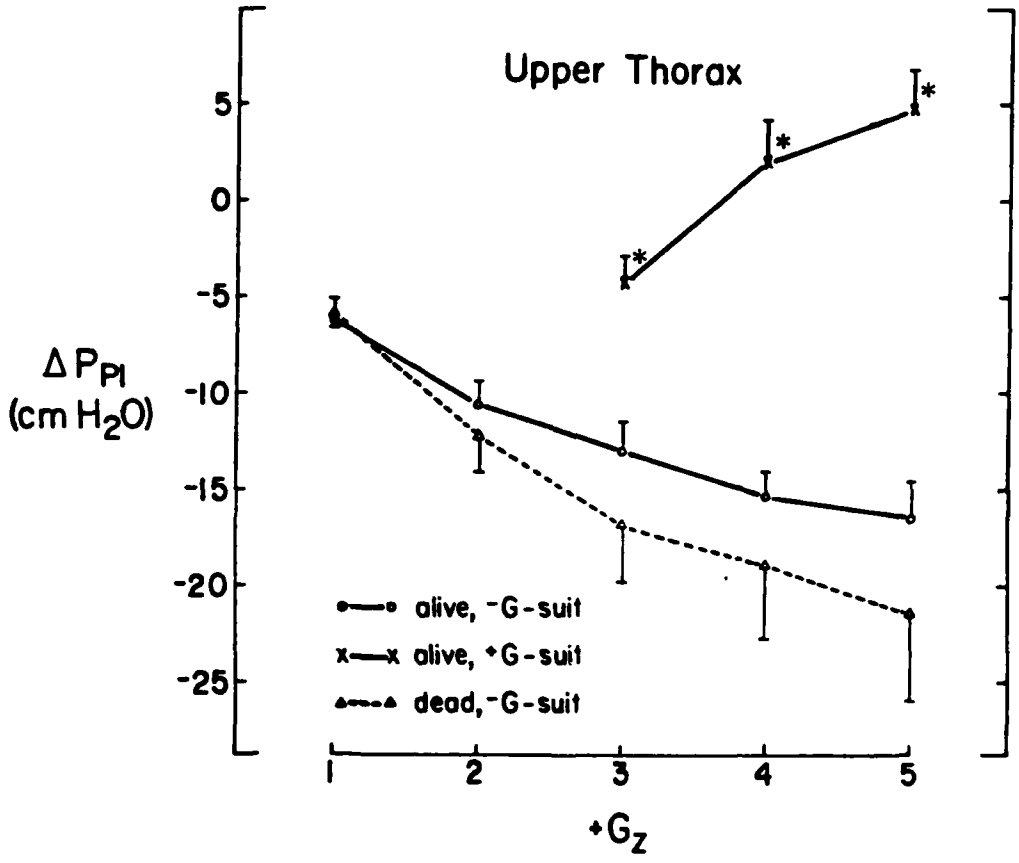

FIGURE I-1. Mean intrapleural pressure changes from 0Gz measured at the third-fourth intercostal space level as a function of +Gz stress in the pig. Three conditions are shown: active muscular tone present (O), active muscular tone present with G-suit abdominal bladder inflation (X), and active muscular tone absent (Δ). Standard errors of the mean are indicated. Statistically significant differences from exposures of the live animal without C-suit (*) were determined by Student's t-test ($P<0.05$).

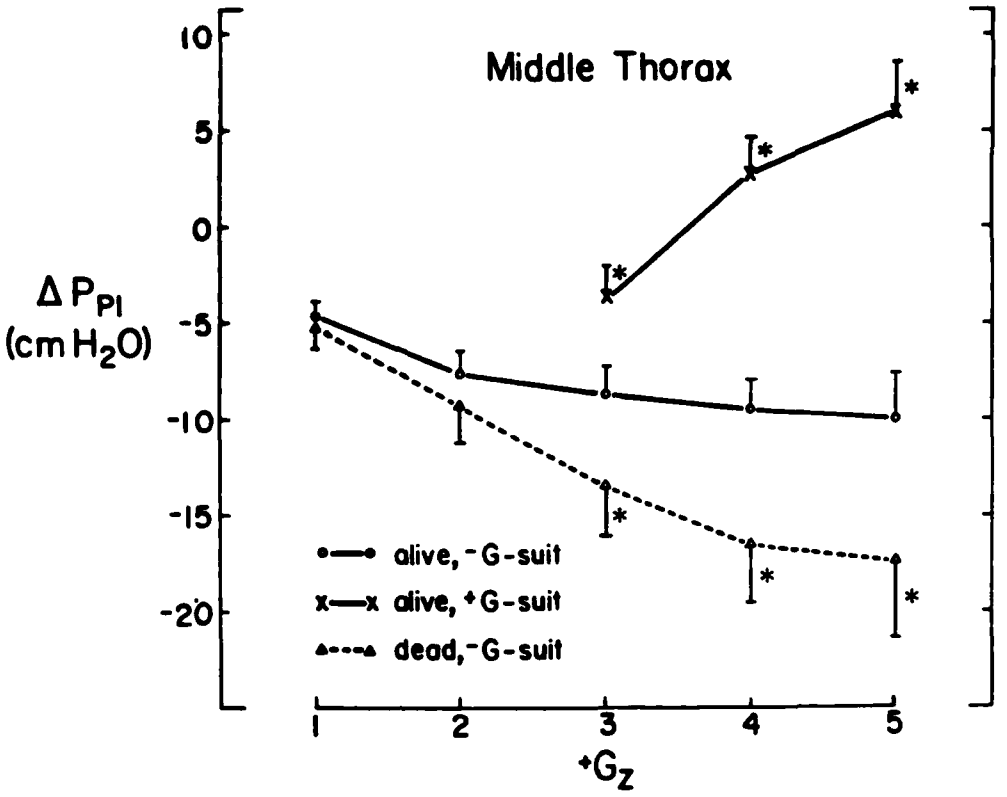

FIGURE I-2. Mean intrapleural pressure changes from 0Gz measured at the fifth-sixth intercostal space level as a function of +Gz stress in the pig. Three conditions are shown: active muscular tone present (O), active muscular tone present with G-suit abdominal bladder inflation (X), and active muscular tone absent (Δ). Standard errors of the mean are indicated. Statistically significant differences from exposures of the live animal without G-suit (*) were determined by Student's t-test ($P<0.05$).

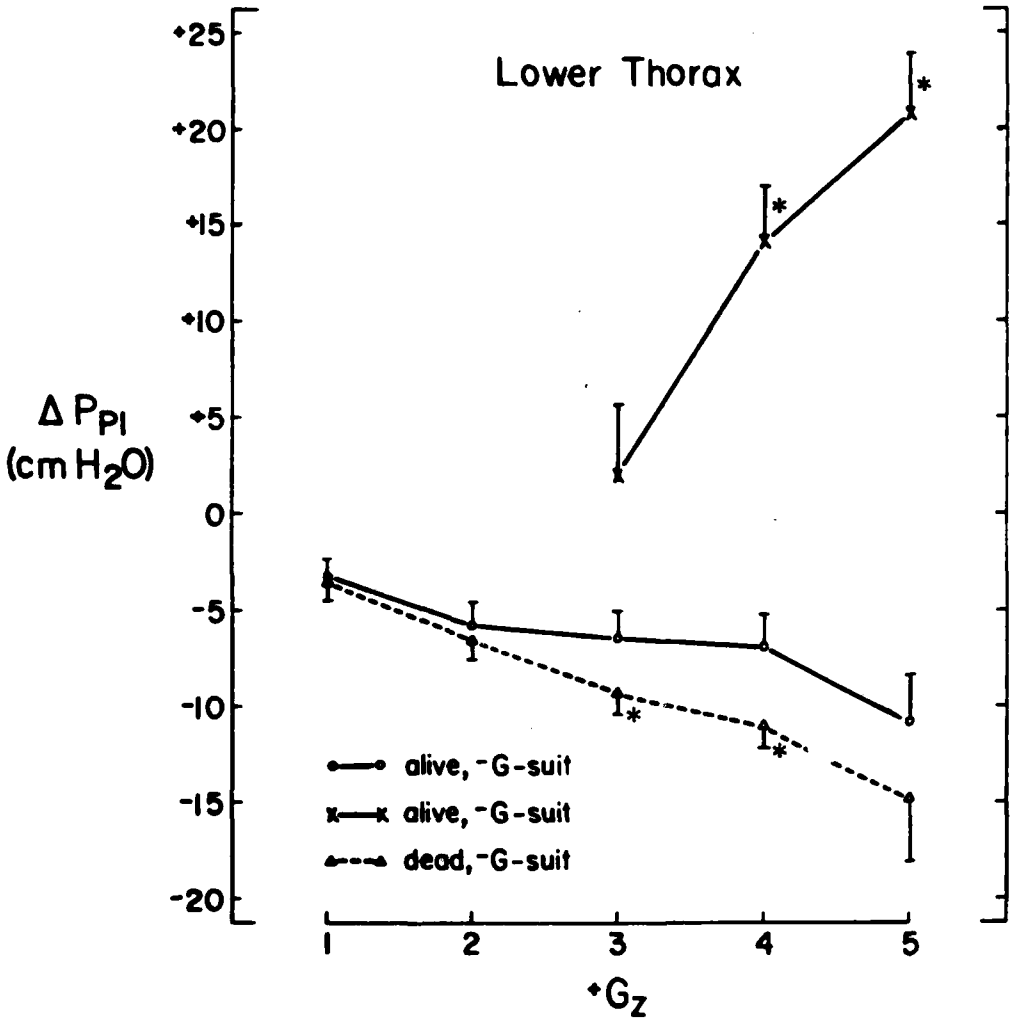

FIGURE I-3. Mean intrapleural pressure changes from 0Gz measured at the eighth-tenth intercostal space level as a function of +Gz stress in the pig. Three conditions are shown: active muscular tone present (O), active muscular tone present with G-suit abdominal bladder inflation (X), and active muscular tone absent (Δ). Standard errors of the mean are indicated. Statistically significant differences from exposures of the live animal without G-suit (*) were determined by Student's t-test ($P<0.05$).

pressure in all lung regions became siginificantly more positive than the control value.

Discussion

Data from the earlier dog studies are compared to corresponding data from the pig in Figures I-4, I-5 and I-6. At the level of the third-fourth intercostal space (Fig. I-4) and the level of the fifth-sixth intercostal space (Fig. I-5), the response to increased +Gz stress is qualitatively similar in the two species. It is interesting to note, however, that, at the level of the third-fourth intercostal space the magnitude of the change in intrapleural pressure appears to be greater in the pig than in the dog. Furthermore, in the non-dependent regions, the pleural pressure changes resulting from imposition of the G-suit appear larger in the pig.

Figure I-6, which shows data for the dog and pig at the eighth-ninth (dog) and eighth-tenth (pig) intercostal spaces raises some interesting questions concerning the influence of chest wall compliance and shape on the intrapleural pressure changes resulting during +Gz stress. The most significant finding in the dog studies was that a region exists at the seventh-eighth intercostal space level where intrapleural pressure remains relatively constant. Above this region, pressure becomes more negative with increasing +Gz stress, while below this region, it becomes more positive. A similar region has been reported to exist in humans exposed to up to +3Gz (Bryan et al., 1966). We were unable to detect an analogous region in the pig.

This region could reflect the transition between a relatively non-compliant upper rib cage and the more compliant lower chest wall. Lupi-Herrera et al. (1976) exposed dogs to negative abdominal pressure

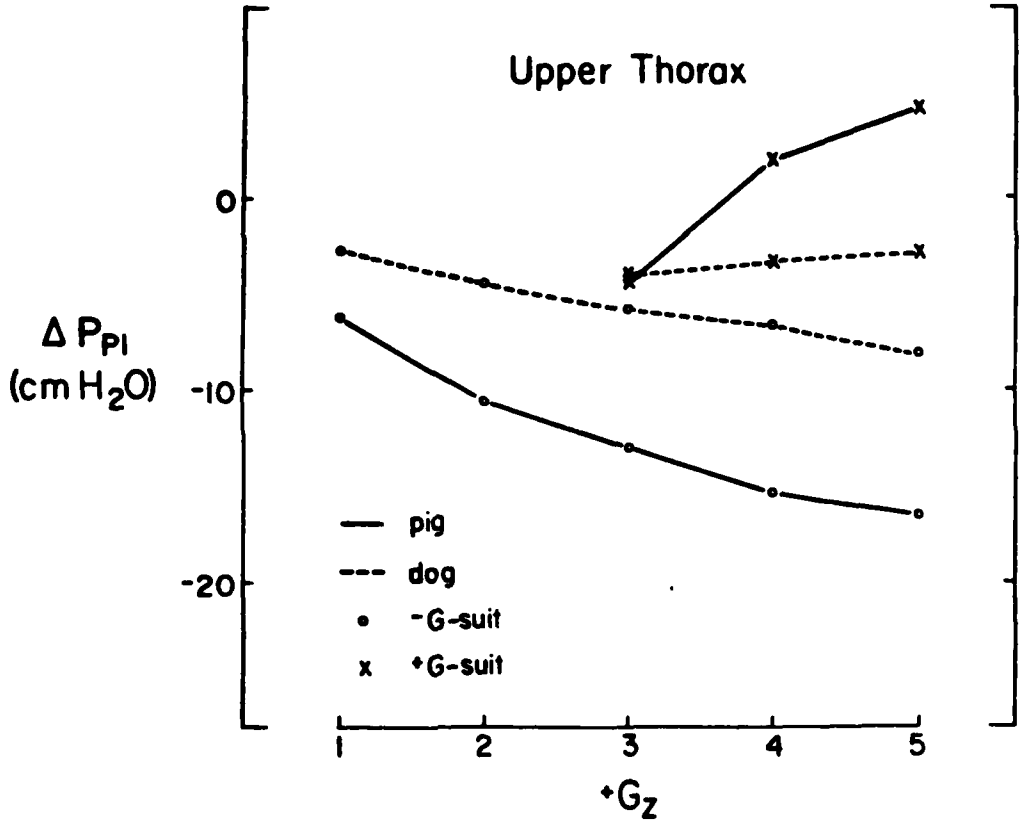

FIGURE I-4. Mean intrapleural pressure changes from 0Gz measured at the third-fourth intercostal space level as a function of +Gz stress in the pig (solid line) and dog (dashed line). Two conditions are shown: Live animals without G-suit inflation (O) and Live animals with G-suit inflation (X).

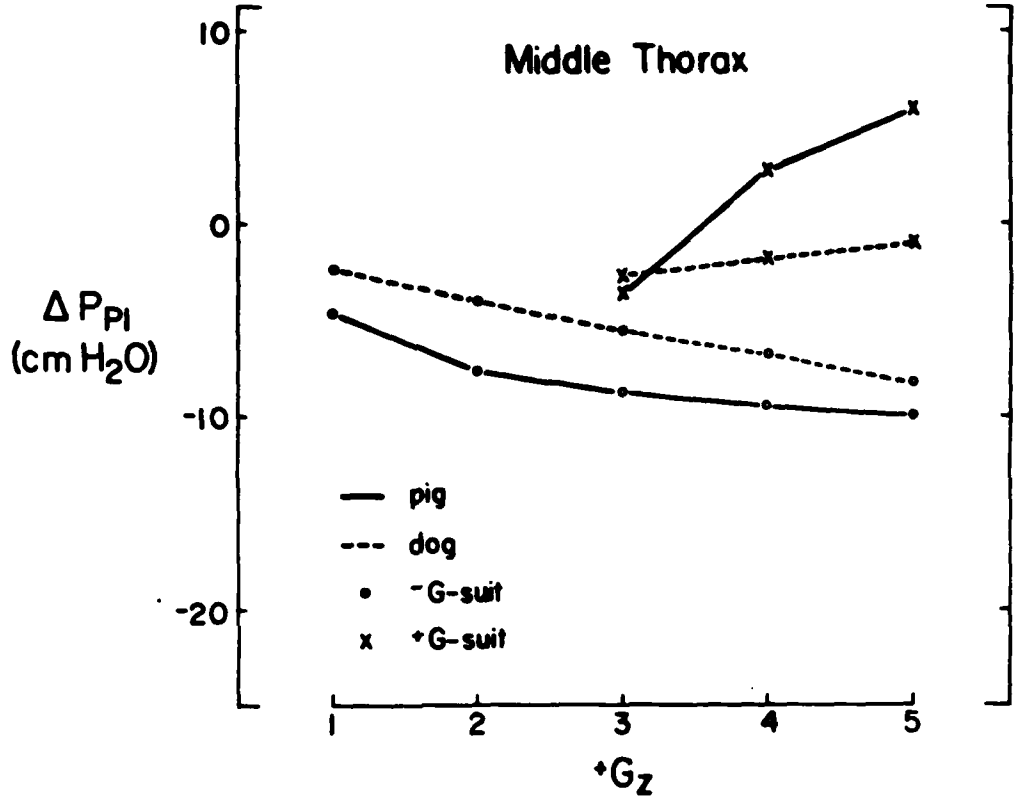

FIGURE I-5. Mean intrapleural pressure changes from 0Gz measured at the fifth-sixth intercostal space level as a function of +Gz stress in the pig (solid line) and dog (dashed line). Two conditions are shown: Live animals without G-suit inflation (0) and Live animals with G-suit inflation (X).

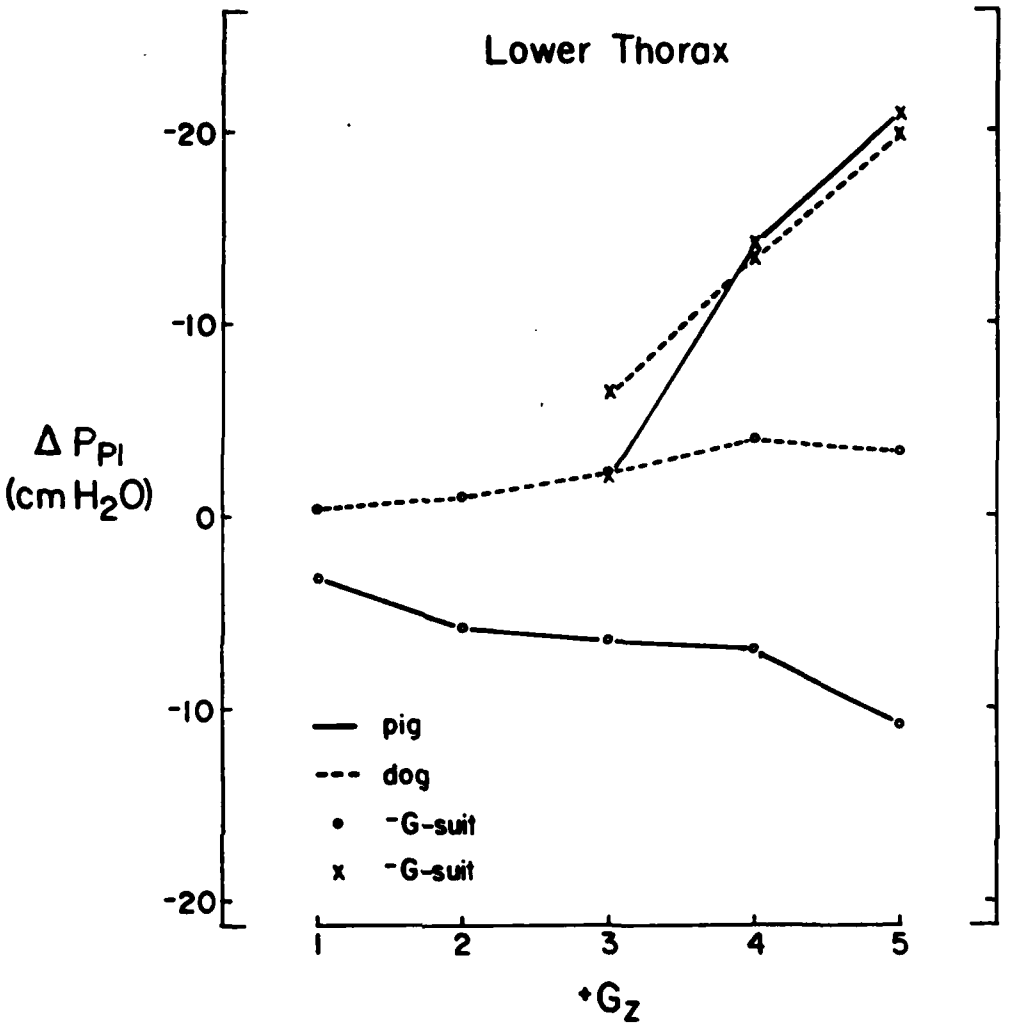

FIGURE I-6. Mean intrapleural pressure changes from 0Gz measured as a function of +Gz stress in the pig (solid line) and dog (dashed line). Pig data represents eighth-ten intercostal space level. Dog data represents eighth-nine intercostal space level. Two conditions are shown: Live animals without G-suit inflation (O) and Live animals with G-suit inflation (X).

and measured the transverse diameter of the thorax at locations corresponding to about the fourth intercostal space and the eight or ninth intercostal space. They reported that the lower rib cage cross-sectional area decreased by 19% whereas the upper cross-sectional area decreased by only 5%. Data from other investigators also indicate that, in the dog, the lower chest wall is considerably more compliant than the upper chest wall regions (D'Angelo et al., 1973; Glazier et al., 1967). We postulated that, when +Gz stress is applied to the system, the lung is pulled caudally, but the upper rib cage remains relatively fixed. A more negative intrapleural pressure is created in this region, and regional volume increases. In the dog, the ribs below the sternum move more freely, and when the thoracic and abdominal contents are pulled in a caudal direction, the lower ribs tend to be pulled inward. The net result is a smaller "container" into which the lung displaced from the upper thorax must fit, and regional intrapleural pressure becomes more positive.

The pig's chest wall is less compliant than the dog's (Attinger and Cahill, 1960). Furthermore, the pig's rib cage appears to be a more rigid container than that of the dog. Hence, minimal displacement of the lower rib cage relative to that seen in the dog would be expected in the pig exposed to +Gz stress. If this is the case, the pig's chest wall should behave more like a container with rigid walls and a compliant lower limit (diaphragm). The above hypothesis would predict that, in such a system, +Gz stress would cause the lung to be pulled caudally, but, with the exception of the lower limit, the volume of the "container" into which the lung is displaced would remain relatively fixed. Thus, the point at which regional intrapleural pressure becomes

positive would reflect the degree to which the relative downward displacement of lung tissue is accommodated by an increase in "container" volume due to the downward movement of the lower boundary (i.e., the diaphragm). The data shown in Figures I-1, I-2 and I-3 indicating that regional intrapleural pressure in the live animal without C-suit continued to become more negative with increasing +Gz stress, even as low as the tenth intercostal space, are consistent with this prediction. If the degree of diaphragm displacement in the pig approximates the relative lung tissue displacement, a positive regional intrapleural pressure may only be seen close to the level of the diaphragm.

The data obtained in the dead animals (Figs. I-1, I-2, I-3) are also consistent with this explanation. Since diaphragm displacement caudally depends to some extent on abdominal muscle tone, removal of abdominal muscle tone would permit further elongation of the lung "container", and regional intrapleural pressure would be expected to become more negative than when abdominal muscular tone was present.

The comparable increase in intrapleural pressure in dog and pig in the dependent regions (Fig. I-6) with G-suit use indicates that, in these anesthetized pigs, the straining maneuver which accompanies G-suit application in awake pigs (Burton, 1973) was not present. The observation that regional intrapleural pressure in non-dependent regions increases less in response to G-suit abdominal bladder inflation in the dog compared to the response in the pig (Fig. I-4, I-5) can be explained on the basis of chest wall compliance. As the C-suit inflates, abdominal compression occurs, and regional intrathoracic pressure in dependent regions increases. As this pressure is transmitted to non-dependent regions in the dog, the relatively

compliant chest wall can increase in volume. In the pig, however, the less compliant chest wall does not move as readily, and pressure remains high.

What are the implications of these data with respect to man exposed to high +Gz stress? The answer to this question is intimately tied to man's use of the M-1 straining maneuver. Bryan et al. (1966) identified an isovolume point in man exposed to up to +3Gz. The subjects in that study did not perform straining maneuvers nor were G-suits used at the low levels of G-stress examined. This finding indicates that the human chest wall can deform in a manner similar to that seen in the dog. However, if an M-1 straining maneuver is made, the chest wall is made less compliant, and the characteristics of the lung-chest wall interaction may approach those seen in the pig. If this is the case, a successful M-1 maneuver may serve to protect the lung from increases in regional intrapleural pressure during +Gz stress and thereby reduce the degree to which airway closure may occur.

Our data (Fig. I-4, I-5) suggest that application of the G-suit abdominal bladder in man without an accompanying M-1 maneuver would result in a larger gas exchange detriment than that expected in the dog and less than that expected in the pig. In the presence of a straining maneuver, however, less of the pressure exerted by the abdominal bladder would be transmitted to the thorax because of increased rigidity of the abdominal wall, and any gas exchange detriment associated with the G-suit would be reduced.

II. AN INEXPENSIVE ASSIST/CONTROL, VOLUME LIMITED ANIMAL VENTILATOR

Most commercially available volume-limited animal ventilators are piston pumps designed for use in situations in which tidal volume and frequency are controlled. Ventilators designed for clinical use, however, provide an additional mode whereby the patient may initiate a breath by creating a negative airway pressure (assist mode). The cost of such machines is prohibitive for use on a limited laboratory basis, and, in some cases, safety measures and other design features incorporated into a machine intended for clinical use may prevent use of the ventilator for specific experimental animal protocols. To circumvent these problems, we modified a field resuscitator so that it could function as a volume-limited ventilator in which animals ranging in size from approximately 5 to 45 kg body weight can be ventilated in controlled or assisted modes. The cost of the completed unit was under $200.

A Globe Safety Products Model 3000 field resuscitator obtained from Federal Surplus Property served as the heart of the ventilator. As originally designed, this unit consists of a pneumatically driven reciprocating bellows governed by a pneumatic control circuit. In this configuration, the unit operates only in a controlled ventilation mode with tidal volume, inspiratory flow rate, expiratory time and, hence, respiratory rate at preset values.

The original pneumatic control circuit was modified to include two electronic timing circuits. A schematic diagram of the reconfigured system is shown in Figure II-1. The bellows excursion is limited at the bottom by an adjustable mechanical stop, thereby allowing adjustment of tidal volume from approximately 75 to 700 ml. Bellows excursion is

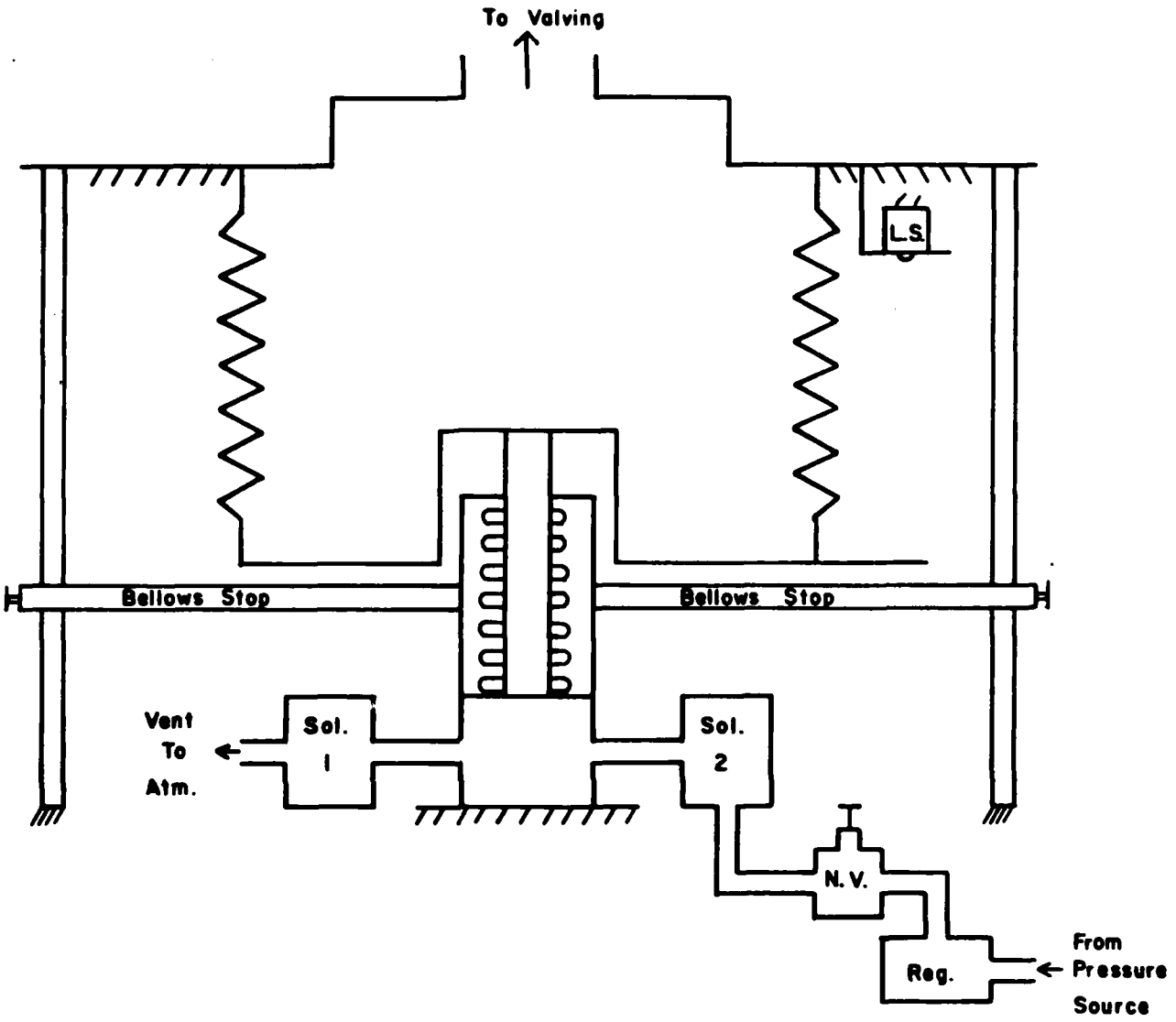

FIGURE II-1. Schematic representation of reconfigured field resuscitator unit. Gas providing pressure for activating bellows movements is regulated to 28 psi (Reg.). The gas passes through a needle valve (N.V.) allowing control of inspiratory flow rate, and it is admitted to the bellows driving chamber through a solenoid (Sol. 2) activated by the electronic control circuit. The end of inspiration is marked by activation of an electronic limit switch (L.S.) which triggers the exhaust solenoid (Sol. 1) and the appropriate timing circuit (see text).

limited at the top by an electronic limit switch. This switch triggers a solenoid exhaust valve on the bellows driving cylinder allowing the bellows to refill from the atmosphere, and it triggers two timers, one for each mode. In the controlled ventilation mode, the governing timer determines expiratory time and is adjustable from 2 to 12 seconds. In the assisted ventilation mode, a 15 second timer is set, and a signal from a pressure transducer measuring airway pressure is fed into the timer circuit. If the signal from this transducer changes polarity, the 15 second timer is overridden ending expiration. The airway pressure at which the unit triggers is adjustable by changing the base line voltage of the amplifier to which the transducer is connected. If an inspiratory effort is not signalled, the 15 second timer marks the end of expiration. When the appropriate timer signals the end of expiration, the exhaust solenoid is closed, and the supply solenoid is opened allowing pressure (28 psi) to build up in the bellows driving cylinder. The gas generating the bellows driving pressure flows through an adjustable needle valve, thereby providing a mechanism for adjusting the rate of bellows movement and, hence, inspiratory flow rate. During expiration, the downward excursion of the bellows is enhanced by a spring mechanism present in the original design.

Driving pressure for the bellows may be supplied from any pressure source developing pressures greater than 28 psi. A pressure regulator reducing the input pressure to 28 psi is incorporated into the design prior to the needle valve.

Valving for the ventilator-patient circuit (Fig. II-2) is accomplished by two passive valves and the non-rebreathing valve that is part of the original ventilator design. A mushroom valve interposed

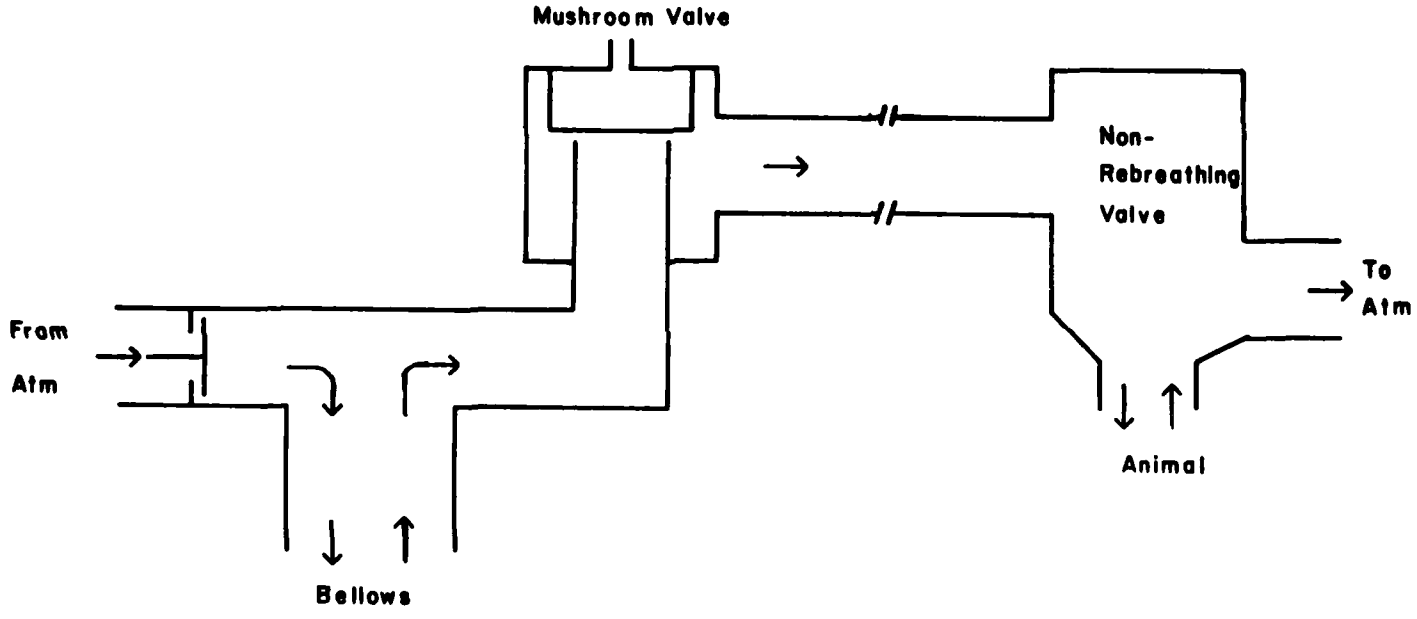

FIGURE II-2. Schematic representation of the valving arrangement used with the modified ventilator (see text).

between the bellows and the non-rebreathing valve on the inspiratory line prevents the animal from breathing "through" the ventilator, as is the case in the original design. Elimination of this clinical safety feature allows the animal to develop a negative airway pressure. The mushroom valve is operated as a passive valve taking advantage of the housing design rather than providing pressure to inflate the "mushroom" balloon to close the valve.

III. INFLUENCE OF THE CHEST WALL ON GAS EXCHANGE DURING MECHANICAL VENTILATION IN DOGS.

The influence of alterations in chest wall motion on gas exchange has not been defined clearly. Minh and his colleagues (1974a, 1974b) examined the pleural pressure gradient and gas exchange in dogs during unilateral electrophrenic stimulation. These investigators successfully altered the pleural pressure gradient and observed an increase in arterial oxygen tension. Schmid et al. (1980) compared chest wall motion and distribution of ventilation in anesthetized supine dogs breathing spontaneously and being mechanically ventilated after muscle paralysis. Although these investigators observed significant differences in abdominal and thoracic movement, they were unable to demonstrate significant differences in the topographical distribution of ventilation between the two states. This study was designed to determine if, during mechanical ventilation, gas exchange is influenced by a muscular effort coordinated with inspiratory flow.

Methods

Five mongrel dogs weighing 19.9 $\pm$ 3.86 Kg were anesthetized with 30 mg/kg pentobarbital sodium administered intravenously and intubated with a cuffed endotracheal tube. A 7 Fr thermal dilution Swan-Ganz catheter was introduced into the right external jugular vein and positioned so that its distal port was in the pulmonary artery and its proximal port was in the right atrium. The femoral artery and vein were cannulated for arterial blood pressure monitoring, arterial blood sampling, and administration of supplemental anesthesia.

The animal was then placed either supine or in a lateral position (right side down), and assisted ventilation was begun with a tidal volume of 15 ml/kg using the ventilator described in Section II of this report. The ventilator triggered when the animal developed -2 cm H_2O airway pressure. After 10 minutes of ventilation, minute ventilation was determined by collecting expired gas for 1-2 minutes. Following this determination, mixed expired oxygen and carbon dioxide tensions were measured, arterial and mixed venous blood samples were drawn and iced for blood-gas determinations, and thermal dilution cardiac output determinations were made in duplicate. Another 10 minute control period was then allowed, and the process was repeated until at least two experimental runs were completed.

In three of the five animals, the orientation of the animal was changed from supine to lateral, and the protocol described above was repeated. In the remaining two animals (one supine, one lateral), at least three determinations were made in the assist mode.

After data had been collected in the assist mode, the animal was paralyzed with 20 mg/kg succinylcholine administered intramuscularly, and controlled ventilation was begun. In this mode, tidal volume and inspiratory flow rate were maintained at the levels established for the assisted ventilation mode. Expiratory time was adjusted so that respiratory rate was comparable to that set by the animal in the assist mode. Hence, all ventilator parameters established during assisted ventilation were essentially the same during controlled ventilation.

Data in the controlled mode were collected in the same manner as in the assist mode. In the three animals in which body position was changed, samples were obtained in both supine and lateral positions.

Results

No differences in any of the measured parameters were detected between the lateral and supine positions. Mean arterial blood gas values for the two modes of ventilation are shown in Figure III-1. Arterial oxygen tension was higher ($P<0.01$, paired t-test), and carbon dioxide was lower ($P<0.01$, paired t-test) when an inspiratory muscular effort accompanied inspiration.

Calculated physiological dead space and cardiac output data are presented in Figure III-2. When the animal was paralyzed and ventilated, physiological dead space increased ($P<0.01$, paired t-test) and cardiac output decreased ($P<0.01$, paired t-test). No differences were detected, however, between the fraction of the cardiac output calculated as representing venous admixture for each ventilation mode.

Minute ventilation measurements confirmed that this parameter was the same during assisted and controlled ventilation modes.

Discussion

An apparent controversy exists in the literature concerning the effects of chest wall motion on gas exchange. Sackner and associates (1974) compared the distribution of ventilation in humans during thoracic breathing to that during diaphragmatic breathing. While differences in distribution of ventilation were detected in normal subjects, no differences were detected between the two types of breathing in patients with chronic obstructive lung disease. Schmid and co-workers (1980) examined the same question in dogs during spontaneous breathing and during mechanical ventilation after muscle paralysis. Although changes in chest wall motion were detected, no differences in the distribution of regional ventilation were observed.

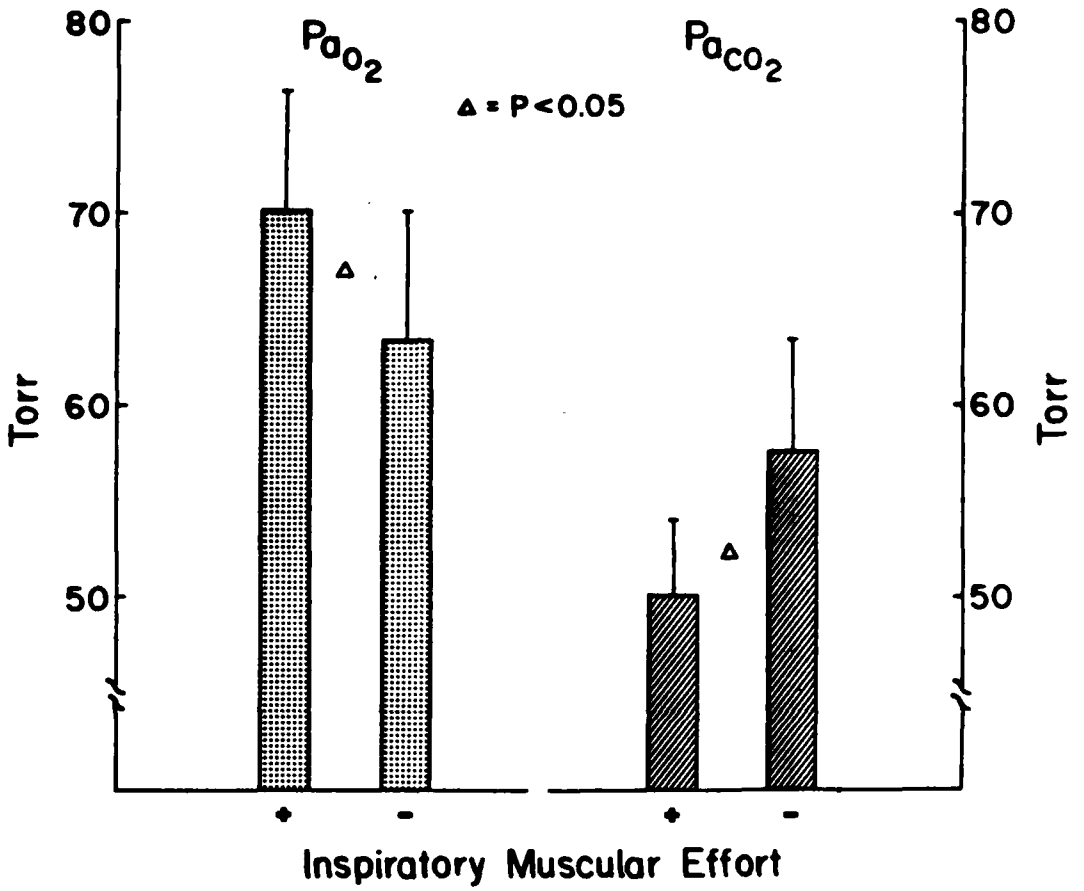

FIGURE III-1. Mean arterial gas tensions obtained with a coordinated inspiratory muscular effort (assisted ventilation) and without a coordinated inspiratory muscular effort (controlled ventilation). Standard error of the mean is indicated. Statistical analysis was performed using a paired t-test.

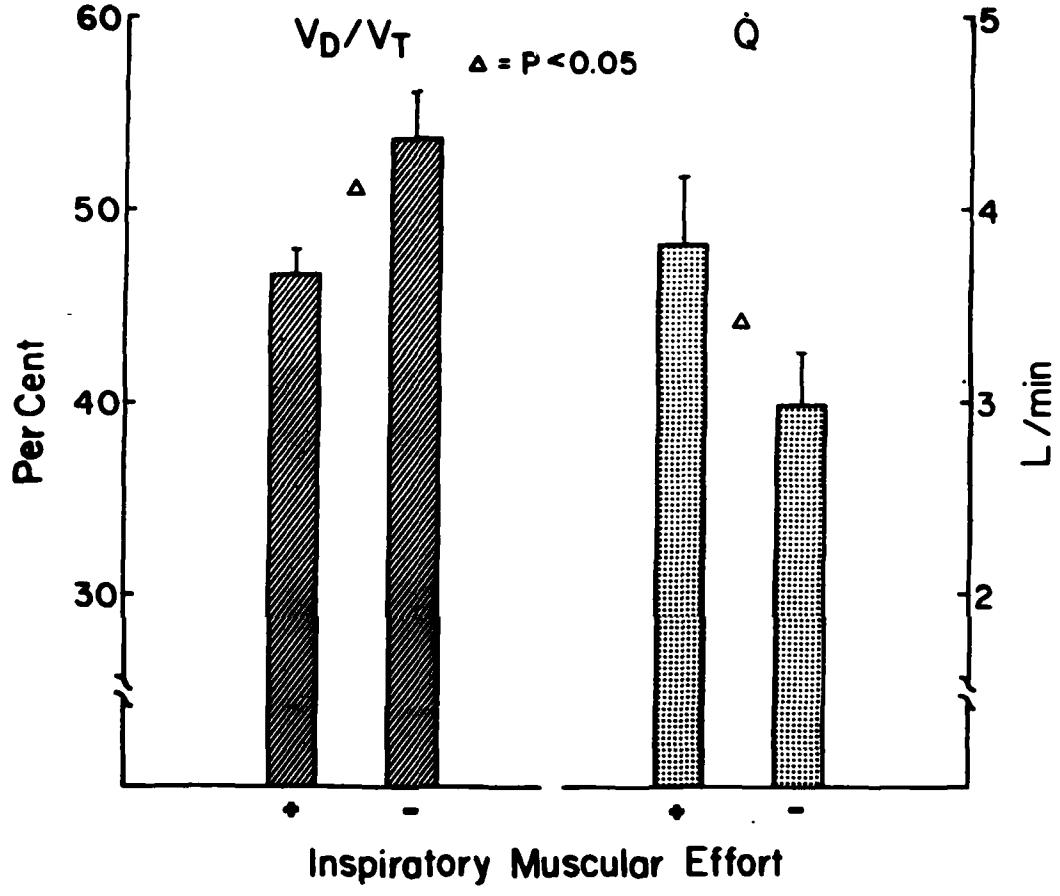

FIGURE III-2. Mean physiological dead space (left panel) and cardiac output (right panel) observed with a coordinated inspiratory muscular effort (assisted ventilation) and without a coordinated inspiratory muscular effort (controlled ventilation). Standard error of the mean is indicated. Statistical analysis was performed using a paired t-test.

Hughes (1979) compared thoracic to abdominal breathing in normal human subjects and concluded that gas exchange was enhanced during abdominal breathing. Minh et al. (1974b) noted increased arterial Po_2 in dogs during right electrophrenic respiration compared to spontaneous breathing.

The implication of these studies is that changes in chest wall motion may not cause significant changes in the distribution of ventilation, but they do alter ventilation-perfusion relationships. In our study, minute ventilation was kept constant, but physiological dead space increased significantly during controllled ventilation (Fig. III-2). The net result of this was a decreased effective alveolar ventilation during controlled ventilation with concommitant changes in gas exchange (Fig. III-1).

Was the increased dead space a result of a shift in ventilation or a shift in perfusion? Rehder et al. (1977) demonstrated changes in the distribution of ventilation when human subjects previously awake, breathing spontaneously were anesthetized, paralyzed and ventilated mechanically. In dogs, however, similar changes have not been demonstrated (Minh et al., 1974b; Schmid et al., 1980). In two additional experiments, we ventilated animals according to the assist-control protocol with Krypton-81m in the inspirate and examined the topographical distribution of ventilation with gamma camera imaging. In these experiments, we were unable to detect gross changes in distribution of ventilation. The difference between human and dog in this respect may reflect the high degree of collateral ventilation in dog lungs relative to normal human lungs (Macklem, 1971). This suggests that the change in dead space resulted from changes in the distribution of perfusion, with perfusion being shifted toward areas having lower

ventilation-perfusion ratios.

Changes in the distribution of perfusion could occur as a result of a decreased cardiac output, alterations in mean intrathoracic pressure or, possibly, regional intrapleural pressure changes resulting from changes in chest wall motion. To get some indication of the Po_2drop expected from a cardiac output fall of the magnitude seen in our data, we analyzed a 3-compartment model of the lung (Modell et al., 1975). Assuming that oxygen consumption remained constant, only about 20% of the observed Po_2drop can be explained on the basis of the cardiac output change alone. In studies aimed at determining the influence of ventilator flow pattern on gas exchange during mechanical ventilation, Modell and Cheney (1979) examined two flow patterns resulting in markedly different mean intrathoracic pressures. In normal dogs, these investigators did not detect changes in gas exchange parameters associated with increased mean intrathoracic pressure. This suggests that local chest wall motion changes can influence distribution of perfusion.

To further examine the mechanism of the ventilation-perfusion distribution changes responsible for gas exchange differences between assisted and controlled ventilation modes, we will be conducting additional experiments in the near future. In these studies, macroaggregated albumen labelled with radioactive technetium will be injected into the pulmonary circulation during the two ventilation modes. For each condition, gamma camera imaging yielding tomographic images will be obtained to assess the topographical distribution of perfusion.

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Modell, H.I. Influence of abdominal restriction on gas exchange during +Gz stress in dogs. The Physiologist 25: 213, 1982 (Abstract).

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Professional personnel associated with research effort

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Michael M. Graham. Assistant Professor, Division of Nuclear Medicine, University of Washington.

Interactions

In the period 1 Apr 1982 to 31 Mar 1983, papers relating to this research effort were presented at the following meetings:

33rd Annual Fall Meeting of the American Physiological Society, 10-15 October 1982, San Diego, California.

Fourth Annual Meeting of the IUPS Commission on Gravitational Physiology, 10-15 October 1982, San Diego, California.